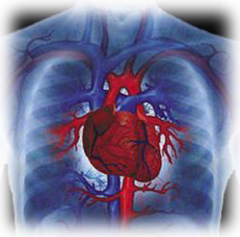
BRADFORD'S HEALTHY HEARTS

NHS
Bradford Districts
Clinical Commissioning Group

Bradford's Healthy Hearts statins FAQ



No.	Questions	Answers	Supporting Evidence
1.	My patient has side effects from statins - what can I do?	Many side effects attributed by patients to statins are in fact not due to statins. The absolute excess risk of the observed harmful unintended effects of statins is very small compared to the beneficial effects of statins on major cardiovascular events. Two recent studies showed 80-92% of patients re-challenged with statins will not have the same aches and pains and will still be taking statins 12 months later[6]. Some were on the same statin, some on different statins. One option is to have a 6-week 'statin holiday' and see if symptoms resolve: The process of atherosclerotic plaque formation is one that occurs over months and years rather than weeks and therefore it is reasonable (in most cases) to suspend statin therapy for a 6 week period - the suspension of statin therapy is not a good idea if there is unstable coronary disease (recent acute coronary syndrome/admission, increasing angina, or angina at rest). **Caution if stopping statins used for high triglycerides – there are risks of pancreatitis if triglycerides climb higher to >20.** There are several choices of statin to try:- 1. Atorvastatin first line.	[6]Discontinuation of Statins in Routine Care Settings: A Cohort Study Zhang H, Plutzky J, Skentzos S et al. *Ann Intern Med.* 2013;158: 526-534. 1) Sikka P et al. Statin intolerance: now a solved problem. J Postgrad Med. 2011 Oct-Dec;57(4):321-8. 2) Matalka MS et al Is alternate daily dose of atorvastatin effective in treating patients with hyperlipidemia? The Alternate Day Versus Daily Dosing of Atorvastatin Study (ADDAS). Am Heart J 2002;144:674-7. 3) Backes JM et al. Effectiveness and tolerability of every-other-day rosuvastatin dosing in patients with prior statin intolerance. Ann Pharmacother 2008;42:341-6 4) Reinhart KM. Strategies to preserve the use of statins in patients with previous muscular adverse

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		Use **Atorvastatin 40mg** OD for **primary** prevention. Use **Atorvastatin 80mg** OD for **secondary** prevention. If intolerant to any dose of atorvastatin, consider: 2. Pravastatin or Simvastatin, then 3. Rosuvastatin. If previously intolerant, try the lowest dose (e.g. atorvastatin 10mg) and then titrate up. Pravastatin is often better tolerated, but is less potent and so is suggested as third line. **"Something" is definitely better than "nothing".** In difficult cases, it is worth trying "intermittent dosing" of statins [1,2,3,4,5]. Start at once weekly dosing of a statin, slowly increasing up to alternate days, then daily, or until side effects start. If doing this, it goes without saying to use the lowest dose of that statin. **Any statin therapy is better than nothing.**	effects.Am J Health Syst Pharm. 2012 Feb 15:69(4):291-300. 5) Mampuya W et al, Treatment strategies in patients with statin intolerance: The Cleveland Clinic experience, American heart journal 1 September 2013, volume 166 issue 3:597-603 **Unintended effects of statins from observational studies in the general population: systematic review and meta-analysis. Macedo et al, BMC Medicine 2014, 12:51**
2.	Are there any contra-indications to Simvastatin?	Be aware of MHRA guidance on simvastatin + amlodipine / diltiazem / verapamil / amiodarone: **Switch to atorvastatin**. MHRA also advise against simvastatin 80mg due to an increased risk of myopathy. **Switch to atorvastatin 40mg od for primary prevention and atorvastatin 80mg od for secondary prevention.**	
3.	Myopathy and asymptomatic raised CK	If statins cause muscle ache, check CK. Myositis is associated with painful muscles AND CK elevation. (see above) Advise a "statin holiday" for up to 6weeks (unless unstable CVD) until	Based in part on: Statin induced myopathy

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		muscle pain resolves and offer trial of alternative statin at low dose with up titration. • If CK < 3x ULN, can continue statin if minimal /no symptoms with regular monitoring CK. • If CK 3-5x upper normal limit. Treatment could be restarted with careful monitoring if tolerated. • If CK is significantly elevated (5-10x upper normal limit) = myositis, treatment should be stopped and seek urgent specialist advice. • If CK > 10x upper normal limit = rhabdomyolysis, needs urgent admission.	BMJ 2008;337:a2286
4.	My patient is already on Simvastatin - should I switch to Atorvastatin?	If the TC>4 then YES. Atorvastatin is now generic and cost effective and quite a lot more potent than simvastatin dose for dose. e.g. simvastatin 40mg vs atorvastatin 40mg. We have designed S1 protocols for mass switches from Simvastatin to Atorvastatin. Most practices have done these. There is evidence that even if TC<4, switching from simvastatin to a more effective dose of Atorvastatin will reduce CV risk, but it is more resource-efficient to target those with higher lipids first, so we suggest those whose TC>4 first.	PROVE IT-TIMI22
5.	When do I check LFTs for statins and what if they are raised?	BHH guidance is that there is no need to measure LFTs in statin use unless there is a clinical concern. This is in line with the American FDA guidance. NICE suggests baseline LFTs be measured before starting a statin, repeated within 3 months of starting treatment, again at 12 months, but not again unless clinically indicated. Other countries advise no checking of LFTs.	

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		Local advice from our consultant hepatologists is that the risk of raised ALT (transaminitis) is <1% and the risk of acute liver failure is clinically negligible. So, from that point of view it is argued that a baseline ALT is not necessary at all. People with a raised ALT: If less than 3 times the upper limit of normal can continue statin with monitoring. Recheck in 4 weeks, if stable and if still <3 x UNL, continue with periodic LFTs. If ALT 120-200, BTHFT Hepatology advice: Patients with **fatty livers** and pre-existing raised ALT can continue statins as long as ALT < 400.	
6.	I'm on a statin now, so I don't need to worry about diet, exercise, weight and/or smoking, right?	Wrong! Lifestyle factors are as effective as taking a standard medication (more if done well). Smoking is the single biggest modifiable risk factor in CV disease. **In higher risk patients, stopping smoking can reduce risk by up to 50%.**	Comparative effectiveness of exercise and drug interventions on mortality outcomes: metaepidemiological study BMJ 2013;347:f5577 Cigarette Smoking, Cardiovascular Disease, and Stroke Circulation. 1997; 96: 3243-3247
7.	How should we treat patients with possible familial hypercholesterolaemia?	People with possible familial hypercholesterolaemia (TC > 7.5 LDL >4.9 prior to statin therapy) should be considered for specialist review.	
8.	Best time to take statins?	Best to take all statins at night except atorvastatin which can be taken anytime of the day. This is because most of the cholesterol is synthesised at night.	

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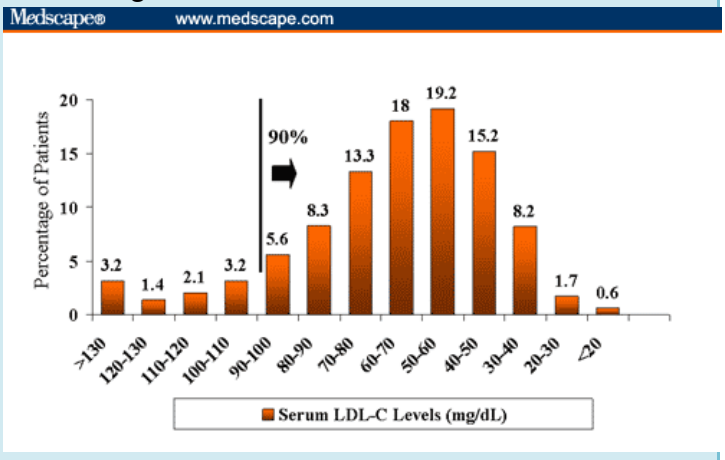
9.	What is the benefit of all these lipid targets?	Cholesterol Treatment Trialists' meta-analysis showed 20% reduction in relative risk for LDL reduction of 1 mmol/L. Full compliance with statin regimens can reduce LDL cholesterol by at least 1·5 mmol/L in many circumstances, and hence might be expected to reduce the incidence of major vascular events by about one third. There is still benefit down to LDL 1.7 (which in practice usually equates to cholesterol well below 4), and safety data down to LDL just over 1! Many practices do not measure LDL or "non-HDL lipids", so a compromise was agreed to target total cholesterol. Please **do** use LDL targets if your practice measures it. Excerpt from BMJ 2014 ;349;g3743: In conclusion, achieving extremely low LDL-C levels appears both safe and effective, and **it does <u>not</u> appear to be necessary to reduce the dose of a statin, if the resultant LDL-C levels fall well below the current guideline recommendations.** This comes from analysis of the PROVE-IT trial. They even had some patients with LDL of <0.5mmol/l. See chart below:	NICE lipid guidance July 2014 BMJ 2014 ;349;g3743

No.	Questions	Answers	Supporting Evidence
		LDL 100mg/dl = 2.59mmol/l (our local units) LDL 50mg/dl = 1.3 mmol/l LDL 20mg/dl = 0.5 mmol/l !  **The data in such groups is smaller and this was a post-hoc analysis, but conclusions were noteworthy in the BMJ paper.** We advise atorvastatin 40mg od for primary prevention and atorvastatin 80mg od for secondary prevention.	

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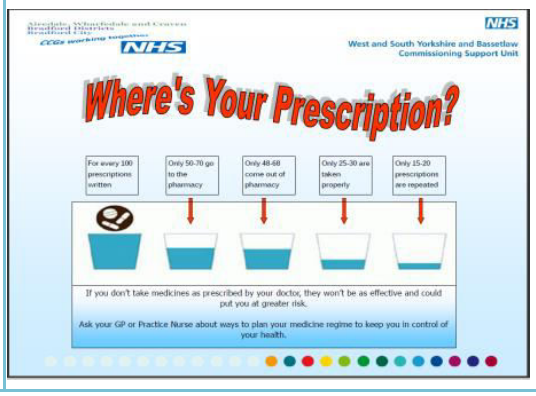
10.	Why Primary Prevention?	Evidence is better for the older we get! Those aged >55 years with an optimal risk factor profile (non-smokers, no diabetes and optimal cholesterol and blood pressure) had much lower lifetime risks. So, optimal primary prevention: • reduced CV events by more than 3-fold • reduced CV death, by 6-fold . reduced coronary heart disease, by 10-fold, compared with those with two or more key risk factors.	Berry JD, Dyer A, Cai X et al. Lifetime risks of cardiovascular disease. N Engl J Med 2012;366:321-9

Adherence & Concordance			***Supporting Evidence***
11.	*Nearly 10% of CVD events due to poor adherence*  	*Patients can reduce risk of event by a quarter by simply taking medicines as prescribed (Choudhury et al. Eur Heart J (2013): 10.1093)* *Tips include:* *Simply explain what each medicine does.* ***Some patients still don't know what their medicines do.*** *Try dose reductions when side effects reported, rather than stopping the drug completely*	

If your question isn't here please email it to Dr Youssef Beaini, Clinical Lead Cardiovascular Disease for CCGs: Youssef.Beaini@Bradford.nhs.uk